Biofilms Part 1: Overcome Candida by Disrupting Its Main Defense

By Stephen F. Olmstead, MD

(This is the first installment of a three-part series about biofilms and their ability to interfere with effective treatment of various diseases. This month’s article discusses Candida biofilms. Next month’s article will address systemic biofilms.)

Fungi are ubiquitous throughout our bodies and the surrounding world.1 The mycobiome is the fungal contribution to the human microbiome, the community of microbes that exists in your body. We know very little about the mycobiome, but the numbers of commensal fungi are certainly orders of magnitude below the more than 100 trillion bacteria inhabiting the human body.2

We have virtually no evidence the mycobiome makes a positive contribution to human health. Fungal cell wall components such as beta-glucan bind receptors on immune cells such as phagocytes, natural killer (NK) cells, and certain classes of T- and B-lymphocytes, so one could hypothesize a beneficial effect on immunity,3 but commensal fungi are almost invariably pathogens or potential pathogens. Organisms such as Cryptococcus neoformans live in low numbers for decades in the lungs of otherwise healthy people only to emerge as a life-threatening fungus when the immune system is suppressed. Small populations of the species-specific fungal pathogen Pneumocystis jirovecii have been found in the mouths of healthy people.

Candida albicans is without question the most significant, potentially pathogenic, commensal fungus encountered by humans.4 C. albicans colonizes the skin and mucosal surfaces of the mouth, genitals, and intestines in 30% to 70% of healthy people at any given time.5 Everyone is likely colonized with C. albicans at some point in life.

Most of the time, Candida colonization is contained and tolerated; no disease ensues. Critical factors that modulate Candida colonization are host immune system responses and the normal microbiota inhabiting the skin and mucosal surfaces. Disruption of normal immune responses and/or composition of the microbiota may lead to C. albicans fungal disease that can be localized, invasive, or systemic. Familiar examples of localized Candida infections are oral thrush and vulvovaginal candidiasis, while candidal esophagitis and disseminated Candida fungemia represent invasive and systemic manifestations.

Our knowledge of how suppressed immunity and antibiotics create conditions for commensal Candida to become pathogenic is limited. Suppressed immunity is associated with oral thrush, while antibiotics increase the risk of vulvovaginal candidiasis, but we do not fully understand why that should be the case.6⁷ Less well appreciated is the ability of C. albicans to cause immune-mediated disease.⁸ Candida has long been associated with allergies, dermatitis, and asthma, and current evidence suggests immune responses to Candida antigens may trigger celiac disease⁹ and inflammatory bowel disease (IBD)¹⁰ in susceptible individuals. A more controversial association is that between C. albicans and multiple symptoms sometimes termed Candida hypersensitivity syndrome, chronic candidiasis sensitivity, or popularly referred to as the “Yeast Connection.”¹¹

HOW CANDIDA EVADES IMMUNE DEFENSES
Candida species are yeasts belonging to the kingdom Fungi, distinguished from plants by a number of characteristics including a lack of photosynthetic capability and the presence of chitin in their cell walls.¹² There are nearly 200 species of Candida, but most cannot replicate at 37°C (98.6°F), so less than a dozen are important in human biology. These include C. albicans, C. glabrata, C. krusei, C. parapsilosis, C. inconspicua, C. dubliniensis, C. guilliermondii, and C. tropicalis.

Candida species alternate between budding yeast cells known as blastospores and filamentous hyphae (a long, branching structure) and pseudohyphae. The ability to transform from blastospore to hyphae and back, known as morphogenesis, is closely linked with virulence.⁴ Morphogenesis is essential to adhesion to cellular and abiotic surfaces as well as to tissue invasion. Filamentous morphogenesis is critical to formation of Candida biofilm,¹³ a gathering of microorganisms firmly attached to a surface.¹⁴,¹⁵ It also facilitates evasion of host immune defenses by modulating immune detection and response.

THE CANDIDA-CELIAC CONNECTION
C. albicans is highly immunogenic and contains up to 178 antigens, with mannoproteins being the most reactive.¹⁶ Candida mannoproteins have been shown to stimulate release of histamine and prostaglandin E₂,¹⁷¹⁸ chemical mediators of allergy and inflammation. Among the more interesting Candida antigens is hyphal wall protein 1 (HWP1). This cell wall protein is highly expressed in hyphae and pseudohyphae and plays a pivotal role in both C. albicans colonization and disease.¹⁹ HWP1 is acted upon by transglutaminase, a cross-linking enzyme that Candida requires to adhere to intestinal cells. Amino acid sequences in HWP1 are similar, in
some instances identical, to those found in gliadin, an allergenic component of wheat responsible for the onset and progression of celiac disease.9

Patients with mucocutaneous candidiasis, a genetic disorder of chronic T cell dysfunction, have been shown to develop elevated antigliadin antibodies in response to HWP1.20 One study found that people with Candida infections and celiac disease had similar, significantly higher anti-HWP1 and antgliadin antibodies than healthy controls.21 Candida colonization is hypothesized to be a trigger for celiac disease, which has inexplicably increased in prevalence over the past decades in tandem with increased antibiotic use and Candida infections.9

CANDIDA’S ROLE IN GI DISORDERS AND OTHER DISEASES

Increased rates of Candida colonization have been observed in both patients suffering from IBD (Crohn’s disease and ulcerative colitis) and their family members.22 Patients with Crohn’s disease have a high prevalence of anti-Saccharomyces cerevisiae antibodies, which are cross-reacting antibodies generated in response to C. albicans colonization.23 An altered immune sensing of Candida gastrointestinal colonization is hypothesized to be associated with the onset of some instances of Crohn’s disease. A high percentage of people with ulcerative colitis are colonized with C. albicans (91%), C. glabrata (7%), or C. inconspicua (2%).24 The addition of the antifungal drug fluconazole or probiotics to conventional mesalamine and azathioprine therapy has been shown to improve remission rates in patients with ulcerative colitis.24 Animal models show that Candida colonization encourages the development of experimental ulcerative colitis after exposure to 2,4,6-trinitrobenezensulfonic acid (TNBS).24 The presence of C. albicans delays healing in TNBS-induced colitis and this can be reversed by fluconazole. High rates of Candida colonization have also been described in peptic ulcer disease and appear to impede healing.24 Human studies show probiotics can promote clearance of Candida and reduce inflammation in the setting of both peptic ulcer disease and IBD.25

Animal research reveals that intestinal Candida colonization intensifies collagen-induced arthritis, a model for rheumatoid arthritis, suggesting a permissive and aggravating role for Candida in autoimmune diseases.26 Intestinal Candida colonization has also been shown in animal models to promote sensitization to food antigens by increasing gut mucosal permeability, or “leaky gut”.27

In humans, intestinal Candida colonization has been linked to allergic asthma, eczema, and hives.9 Neuropsychiatric symptoms are often prominent in patients diagnosed with fungus-related diseases. One possible mechanism deserving of investigation is the cross-reactivity of HWP1 and gliadin, as neuropsychiatric symptoms are also very common in celiac disease.28 These symptoms are thought to be immunologically mediated and among the most common are depression, personality changes, and attention deficit. The evidence for plausible mechanisms of action whereby Candida colonization may cause gastrointestinal and systemic disease is compelling.

USING DIET AND ANTIMUNGLS TO ELIMINATE CANDIDA

An expanding body of human and animal research clearly implicates gastrointestinal, oral, and genital Candida colonization with localized and systemic inflammation, allergies, and autoimmune processes. Nevertheless, the concepts of candidiasis sensitivity and fungus-related disease continue to be criticized in part because of the supposed lack of human studies on the efficacy of diet and antifungal interventions.11 However, burgeoning clinical evidence suggests dietary interventions and antifungal therapy may result in symptomatic improvement in patients with multisystem symptomatic complaints.

Many clinicians have long noted that a subset of patients who meet the diagnostic criteria for chronic fatigue syndrome respond to antifungal therapy and a diet that restricts sugars, starches, and yeast-related foods. In 1989, Jessop presented her experience treating 1,100 such patients at the University of California San Francisco.29 Prominent symptoms she noted in her patients included fatigue, myalgia, headache, depression, dizziness, joint pain, night sweats, morning stiffness, and postexercise malaise. The neurologic examination was abnormal in 30% of patients. Approximately 80% of the subjects had repeated antibiotic courses for a variety of indications. Of the 1,100 patients, 685 were on disability.

Jessop prescribed 200 mg per day of the antifungal ketoconazole along with a diet free of alcohol, added sugar, fruit, and fruit juice. The average length of treatment was five months (range: 3 to 12 months). In follow-up, 84% of patients recovered and only 12 of 685 remained on disability. Jessop concluded that Candida infection with production of an unknown systemic toxin was the cause of the disease. Unfortunately, the study was never published and the interventions were unblinded and uncontrolled.

In 1990, Dismukes and coworkers published a study of the antifungal drug nystatin for candidiasis hypersensitivity syndrome.30 The trial involved 42 women with a history of Candida vaginitis and unexplained polysomatic symptoms. Subjects were randomized to receive oral and vaginal nystatin, oral nystatin alone, vaginal nystatin alone, or placebo. Every eight weeks, participants were crossed over to a different study group so that all subjects received each regimen. Follow-up included clinical assessments, completion of a questionnaire evaluating presence and severity of 18 different symptoms, and the SCL-90-R multidimensional symptom inventory, which focuses on psychiatric complaints. The investigators found that nystatin improved symptoms related to vaginitis, but was no better than placebo at relieving other complaints.

At the same time, the Dismukes study, published in the prestigious New England Journal of Medicine, was widely touted as disproving the concept of candidiasis sensitivity. However, the study had significant limitations. The number of subjects was extremely small, and dividing participants into four intervention groups served to render the trial so vastly underpowered as to make any conclusions meaningless. Further invalidating the findings, crossover designs in antimicrobial studies are widely regarded as inappropriate.31

In 1998, Eaton and Howard published a small, retrospective study of 25 patients diagnosed with fungus-type gut imbalance on the basis of an increase in blood ethanol levels following an oral glucose load.32 All subjects were instructed in a diet that restricted starch and excluded yeast and mold-containing foods. Patients who had not improved after one month received either oral nystatin or amphotericin B. Interventions were continued for a minimum of three months, but usually six months. The investigators found that 21 of 25 patients improved on diet alone and the remainder experienced resolution of symptoms with the antifungal agents. Small numbers, lack of a control group, the absence of blinding, and lack of objective assessment at baseline and follow-up all compromise the findings of this study.

The best designed and conducted study of diet and antifungal interventions for candidiasis sensitivity or fungus-related disease comes from researchers in Norway.16 These investigators carried out a prospective, double-blind, randomized, placebo-controlled trial. They developed a seven-item screening questionnaire termed the Fungus-related Disease Questionnaire-7 (FRDQ-7) derived from a statistical discrimination analysis of the Candida-related Complex (CRC) scheme found in Crook’s The Yeast Connection and the
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**CANDIDA INFECTIONS**

A high score (10 or greater) was used to qualify subjects for entry into the study.

Patients were randomized to oral nystatin or placebo for four weeks. They were also allowed to adhere to their usual diet or to avoid foods containing sugars, yeasts, and molds. Participants were assessed with a 70-item CRC questionnaire evaluating the presence and severity of symptoms. Nystatin was found to be significantly better than placebo in improving symptoms. Diet plus nystatin was significantly better than nystatin alone, and diet alone significantly improved symptoms better than placebo. Remarkably, this well-conducted study has received scant attention and over the past decade no additional clinical trials have been conducted to confirm or refute its findings.

**HOW CANDIDA PROTECTS ITSELF**

A consistent observation by both patients and practitioners is that while select individuals with symptoms may respond to diet and antifungal interventions, the response is usually limited in time and relapse is the rule. One possible explanation is that *C. albicans*, as well as other *Candida* species and fungi in general, reside within biofilm because it affords them protection from competitors, predators, antimicrobial agents, host immune responses, and toxins. Biofilm also prevents microbial communities from being dislodged from an environment and optimizes nutrient utilization. Biofilm formation is an integral part of *Candida* colonization on mucosal surfaces and skin, and transition from blastospore to hyphae is an integral part of biofilm development.

As *Candida* enters into biofilm mode, genes coding for antifungal resistance are expressed. Biofilm existence makes *Candida* highly resistant to eradication, yet allows it to remain highly immunogenic, capable of stimulating host defensive immune responses as well as generating allergic and autoimmune reactions. Immunogenic, colonizing *Candida* biofilm communities offer an explanation for why fungal overgrowth is rarely documented in patients with multiple symptoms and for high relapse rates following treatment. Biofilm also explains in part why mucosal infections such as vulvovaginal candidiasis become recurrent or chronic in nature. Fresh approaches aimed at disrupting *Candida* biofilm formation hold great promise for improving treatment responses in people with localized *Candida* infections as well as those with multiple symptoms of candidiasis sensitivity and fungus-related disease.

**ELIMINATING CHRONIC CANDIDA INFECTIONS**

An approach to *Candida* biofilm eradication involves the use of supplemental enzymes, chelating agents, and probiotics together with naturally occurring antimicrobials and agents that disrupt an essential step in biofilm formation known as quorum sensing.

**ENZYMES**

Enzymes that disrupt the polysaccharide components of biofilm are effective antibiofilm agents. Cellulase is a major component of most biofilms. Cellulase is a group of enzymes that break apart the chemical bonds in cellulose. Cellulase has been shown to significantly reduce biofilm formation. Glucans and chitin are also major components of *Candida* biofilm and cell walls. Combining beta-glucanase and chitinase with cellulase yields an enzyme formulation with powerful antifungal biofilm activity. The addition of a protease-peptidase complex with high DPP-IV activity facilitates breakdown of biofilm matrix protein components as well as degrading cell wall mannoproteins including the immunogenic virulence factor HWP1.

**METAL CHELATING AGENTS**

Metals such as calcium, magnesium, and iron are critical to biofilm formation and maintenance. The disodium salt of ethylenediaminetetraacetic acid (EDTA) is a powerful metal chelator. Disodium EDTA has well established antibiofilm activity mediated by complexing with metals required for cross-linking biofilm matrices. Chelation of calcium is particularly important for the antibiofilm activity of EDTA. Disodium EDTA also causes structural damage to bacterial cell walls making them more permeable to antimicrobial agents. EDTA has been shown to inhibit filamentation and biofilm formation by *C. albicans*. The combination of disodium EDTA with enzymes synergistically disrupts fungal biofilms, potentially facilitating their eradication.

**SACCHAROMYCES BOULARDII**

*S. boulardii*, a variant of *S. cerevisiae*, is a probiotic yeast. It has been used worldwide for over 60 years for a variety of indications such as IBD, prevention of antibiotic-associated diarrhea, treatment of *Clostridium difficile*-associated disease, and treatment of *Blastocystis hominis* and other unicellular parasitic infections. *S. boulardii* has been widely used as an antifungal agent, but its mechanisms of action have been unclear. Recently, *S. boulardii* has been found to significantly inhibit *Candida* adhesion, hyphae growth, and biofilm formation -- effects mediated primarily by capric acid secretion. *S. boulardii’s* ability to disrupt critical aspects necessary for *Candida* colonization and virulence makes it an important addition to the therapeutic toolkit for treating people with fungal-related diseases.

**LACTOBACILLUS PROBIOTICS**

Lactobacillus probiotics have been shown in human studies to promote clearance of *Candida* associated with gastrointestinal inflammatory lesions. In combination with standard therapies, they have been shown to reduce inflammation and accelerate healing in the setting of peptic ulcer disease and IBD.

**PROPOLIS**

Propolis is a complex mix of plant exudates collected by bees that also contains beeswax and bee salivary enzymes. Propolis has been used for millennia as a preservative and medicine for fever, muscle pain, arthritis, and eczema. Roman medical texts discuss the use of propolis for wound care and as an agent that reduces scarring. Propolis has well documented antimicrobial and antibiofilm properties. In the laboratory, propolis extract has demonstrated direct fungicidal effects on *C. albicans* isolated from women with *Candida* vulvovaginitis. Propolis also strongly suppresses *Candida* biofilm formation on abiotic surfaces. Propolis inhibits the morphogenic transition from yeast to hyphae and is an effective antifungal in murine models of vulvovaginal candidiasis.

**CINNAMON BARK EXTRACT**

Cinnamon bark extract has a long history of use in traditional medicines. Cinnamon oil and cinnamaldehyde have broad antimicrobial activities and are lethal to a variety of yeasts and molds. Cinnamaldehyde inhibits morphogenesis and enzyme secretion by *Candida* isolates from the human mouth. Cinnamon essential oil components cinnamaldehyde and eugenol are fungicidal against *Candida* that is resistant to the antifungal drug fluconazole and enhance the lethal effects of antifungals against *Candida* living in biofilm.

**QUERCETIN**

Quercetin is a flavonoid widely found in fruits and leafy vegetables. Quercetin has been studied for its antioxidant, immunomodulating, anti-inflammatory, antineoplastic, antidiabetic, and other properties. Quercetin is a sensitizing agent for fluconazole-resistant *C. albicans*. It promotes fluconazole-mediated cell killing and
apoptotic cell death. In vitro, quercetin strongly suppresses biofilm formation, hyphal development, and production of candidal virulence factors. Quercetin’s mechanism of action appears to be upregulation of farnesol, a quorum sensing molecule that inhibits *Candida* biofilm formation. Quercetin has also been shown to enhance the sensitivity of *C. tropicalis* to fluconazole.56

**ELIMINATING CANDIDA**

If you have had trouble eradicating your *Candida* infection, keep in mind that *Candida* colonization occurs within mucosal and skin biofilms where it is highly resistant to eradication. Enzymes, chelating agents, probiotics including *S. boulardii*, propolis, and botanical compounds represent novel approaches that may render dietary and antifungal treatment of people with *Candida*-associated disorders more successful.


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**Enhancing Neurological Health Part 2: Improving Memory, Attention Span, and IQ**

By Chris D. Meletis, ND and Kimberly Wilkes

[This is the second installment in a four-part series about improving your brain health and memory. In January’s eNews, Dr. Meletis discussed Parkinson’s disease. Future installments will cover Alzheimer’s disease and depression.]

Peak mental performance is an advantage while attending school, increasing productivity, excelling in your chosen career, and during aging to maintain independence. Research indicates a number of factors can either impair memory in the elderly or prevent middle-aged individuals, young adults, and children from functioning at their peak mental capacity. Research also demonstrates there are specific ways an individual’s intelligence quotient (IQ) can be raised.

This article will discuss how you can improve your memory and attention span—no matter what your age. It will also describe factors involved in memory impairment during aging. Because the next article in this series will address Alzheimer’s disease, the focus of this article will remain on general cognitive health and normal, age-related cognitive decline. Applying the cognitive-enhancing principles outlined in this article, however, may also help you sustain healthy memory and intellect as you age.

**IS AGE-RELATED MEMORY IMPAIRMENT INEVITABLE?**

Even in elderly patients who do not have dementia, a decline in cognitive abilities can have negative health consequences. Researchers studied 1,037 elderly subjects who did not have dementia, and after controlling for depression and diseases related to cognitive decline, found that normal, age-related cognitive impairment predicts mortality.1 For most people, aging is associated with a decline in cognitive function including reduced processing speed, memory, motor performance, white matter integrity, brain volume, and executive function (a term used to describe your capacity to carry out cognitive processes such as working memory, reasoning, flexibility, problem solving, planning, and execution).2-4 However, not every elderly person experiences cognitive decline, suggesting memory impairment with aging isn’t inevitable.2 One group of researchers named elderly individuals who retain their cognitive abilities throughout their life “Cognitive SuperAgers.”
These researchers studied persons over the age of 80 whose episodic memory was as good or better than that of 50- to 65-year-old subjects and whose scores in other measures of cognition were average for their age or better, and compared these SuperAgers to healthy, age- and education-matched controls.5

The researchers found that the cerebral cortex of the brain in SuperAgers was markedly thicker than that of the controls, who demonstrated cortical atrophy characteristic of persons in this age group. There was no evidence of atrophy in the cerebral cortex of the SuperAgers. The study did not reveal whether SuperAgers had an especially thick cortex from birth or whether the cortex had experienced less atrophy over time. The thickness of a region of the brain known as the left anterior cingulate cortex was also significantly greater in SuperAgers compared to both elderly and middle-aged controls. Research has linked abnormalities in the cingulate cortex to the first stages of Alzheimer’s disease,6 suggesting support and maintenance of this area while aging might be advantageous to cognitive preservation.

**EPIGENETIC CHANGES**

Epigenetics are genetic changes triggered by external or environmental factors that do not involve alterations in the DNA sequence. Two epigenetic mechanisms involved in learning and memory are histone acetylation and DNA methylation.7 Histone acetylation is a powerful mechanism to modulate the genes involved in learning and memory.8-10 Age-related alterations in DNA methylation may also impair memory.11 Evidence suggests DNA methylation decreases globally with age, but may increase locally across multiple brain regions,12,13 adversely affecting cognitive function during aging.14

**YOUR BRAIN ON STRESS**

Chronic stress can impair cognitive function in both younger and older adults. Researchers have studied the effects of job strain, psychological demands, and job control on cognitive impairment. One group investigated whether 1,429 subjects participating in the Framingham Offspring Study were experiencing job stress at the beginning of the study and administered neuropsychological assessments approximately 15 and 21 years afterwards. Increased workplace stress combined with having a low amount of control over work situations correlated with a reduction in verbal learning and memory. Word recognition skills also declined as a result of job stress.15

Another group of researchers investigated the link between perceived stress and cognitive decline in 116 adults aged 67 to 96. The researchers measured cognitive function and perceived stress every six months over a two-year period. The higher the amount of stress the subjects reported, the greater the decline in performance on tasks of attention, working memory, and speed of processing. Increases in perceived stress correlated with cognitive decline over time.16

**INSOMNIA AND MEMORY**

Sleep quality affects cognitive function, especially sustained attention, vigilance, memory, emotional processing, and working memory. Scientists performed functional MRIs in 25 patients with primary insomnia and 25 patients without insomnia. Patients with primary insomnia had the same level of cognitive performance as the individuals without insomnia. However, patients with insomnia demonstrated weaker activity of task-related working memory regions of the brain. Furthermore, people with insomnia had abnormal function of their task-irrelevant brain areas during tasks that required working memory.17 Other researchers noted a decline in performance after multiple instances of partial sleep loss or a single instance of total sleep deprivation in the study subjects. Having endured partial sleep deprivation on other occasions was associated with weaker cognitive ability when subjects were totally deprived of sleep, an effect observed predominantly in the early morning. The two areas of cognitive function most severely affected in both partial and total sleep deprivation were subjective alertness and sustained attention.18

While sleep deprivation impairs cognitive function, a daytime nap can improve cognitive performance even in individuals who are not sleep deprived. Evidence indicates that naps can enhance both conscious19 and unconscious memory,20 as well as emotional processing.21 Researchers studied the effect of a daytime nap on working memory in 80 healthy college students aged 17 to 23. The investigators randomized the students into two groups. One group was allowed to take daytime naps and the other instructed to stay awake. The subjects completed a sleep diary and state-measurements of sleepiness as well as a psychomotor vigilance test and a working memory task at the beginning and end of the nap and wake sessions. Subjects who took naps performed more accurately on the working memory task and had fewer lapses on the psychomotor vigilance test. Accuracy on the working memory task was associated with the length of rapid eye movement (REM) sleep and the amount of sleep received while napping.22

**OBESITY’S EFFECT ON COGNITION**

Obesity impairs vascular function and can increase the risk of dementia. Researchers observed that young and middle-aged adults who have a higher body mass index (BMI) experience greater cognitive impairment as they age.23 Other scientists measured attention and executive function in 408 healthy subjects from 20 to 82 years of age.24 They observed that the higher the BMI, the worse the results on cognitive tests. Overweight and obese subjects with a BMI greater than 25 performed worse on tests of executive function compared to normal-weight adults with a BMI from 18.5 to 24.9.

In this study, attention was not affected by body weight and there was no indication that the effect of BMI on cognitive function differed with age. The association between obesity and cognitive decline was independent of obesity-caused diseases that are in themselves related to cognitive impairment.25 Interestingly, another study of 66 community-dwelling, older adults aged 70 to 80 found that developing obesity later in life had a protective effect on cognition.26 This paradoxical effect has yet to be explained.

**HOW HORMONES AFFECT YOUR COGNITIVE FUNCTION**

Evidence from preclinical and clinical studies suggests steroid hormones play an important role in brain health. Cognitive impairment often accompanies menopause, a time associated with low estrogen levels. Estrogen replacement therapy, on the other hand, is associated with improved memory and has been shown to reverse markers of age-related cognitive decline in the hippocampus of animals.27

Estrogen’s capacity to improve cognitive function during menopause may involve its effects on the cholinergic system. Cholinergic deficits have been implicated in age-related memory decline and Alzheimer’s disease. A study of postmenopausal women showed that women using estrogen replacement therapy had a higher density of cholinergic receptors compared to women who had never used estrogen therapy. Estrogen therapy users also demonstrated improved executive function compared to nonusers.28

Recent randomized, placebo-controlled, clinical trials indicate estrogen replacement therapy is most effective when started at the
replacement studies used bioidentical estrogens, which have a better safety profile than synthetic conjugated estrogens.

Pregnenolone is another hormone that influences cognitive health. In mice, pregnenolone has been shown to prevent ethanol-induced memory impairment. Furthermore, researchers found that administration to male mice of pregnenolone, pregnenolone sulfate, dehydroepiandrosterone (DHEA), dehydroepiandrosterone sulfate (DHEAS), androstenedione, testosterone, dihydrotestosterone, or aldosterone all improved the rodents’ memory as tested by their ability to avoid foot shocks. Estrone, estradiol, progesterone, or 16-beta-bromoepiandrosterone had no effect on the ability of the mice to avoid foot shocks. Of all the hormones tested, pregnenolone and pregnenolone sulfate had the greatest effectiveness.

In another study, researchers observed a significant improvement in the synaptic plasticity of brain areas involved in memory in aged rats injected with pregnenolone. Sympathetic plasticity refers to the ability of synapses to change over time and is related to learning and memory. Animal studies also indicate progesterone administration may play a role in learning and memory.

### LOW THYROID EQUALS REDUCED COGNITIVE FUNCTION

Hypothyroidism is known to impair cognitive function. Children with congenital hypothyroidism show evidence of decreased IQ compared to healthy controls. Adults with lower thyroid stimulating hormone (TSH) levels (an indicator of low thyroid function) experience a more rapid decline over time in working memory and visuospatial function (the ability to process and interpret visual information about where objects are located). An analysis of patients with normal thyroid function found that women with high-normal TSH concentrations might be at higher risk of cognitive decline. Group comparisons showed significant differences in the immediate recall portion of the verbal memory task in women with higher TSH.

Other studies have yielded similar results. In one study, severe hypothyroidism was associated with a delay in fine-motor performance of hands and reaction times in emergency braking tests. Braking times while driving were 8.5% greater in hypothyroid patients, which equals the effect of driving with a blood alcohol level of 0.082 g/100 mL, an amount exceeding the U.S. legal driving limit. Subjects with hypothyroidism also exhibited declines in executive tests. In contrast, episodic memory and learning retrieval functions were not adversely affected.

### OTHER FACTORS INVOLVED IN BRAIN HEALTH

Hydration can affect cognitive performance, especially in children and the elderly.

Hearing loss in the elderly also affects working memory, which is important for understanding speech in noisy and distracting surroundings. On the other hand, hearing aids have been shown to improve working memory.

Another factor that can interfere with cognitive function is an electrolyte disorder known as hyponatremia, a low-sodium state that has been associated with cognitive dysfunction and attention problems. Rat models show hyponatremia is associated with gait disturbances, cognitive dysfunction, and abnormalities in long-term enhancement of specific synapses in the hippocampus of the brain.

In addition, high levels of the non-protein amino acid homocysteine and dysfunction in the powerhouse of cells known as mitochondria can influence brain health over time. These two factors will be discussed in depth in the next part of this article series, which will address Alzheimer’s disease.

### ENHANCING COGNITIVE HEALTH

Cognitive health at all ages can be enhanced through the use of lifestyle measures and supplementation with botanicals and nutrients that help protect the brain.

### PHYSICAL ACTIVITY AND YOGA

Exercise has beneficial effects on cognition. In rats, exercise has been shown to improve memory and reduce the hippocampal damage resulting from chronic stress. Abundant evidence indicates exercise can enhance cognitive function in the elderly. In a review of 27 studies, 26 found a positive association between exercise and either preservation or improvement of cognitive abilities in subjects over 60 years of age. A study published after the review investigated the effect of resistance exercise on spatial awareness (understanding how objects relate to each other and to the observer) and visual and motor function in seniors. The authors concluded that resistance exercise training was “likely beneficial” in enhancing spatial awareness and visual reaction times. There was a 40% improvement in spatial awareness while visual reaction time was enhanced by 14.6%. No changes occurred in motor reaction time.

There is also evidence that exercise improves cognitive function in healthy, young-to-middle-aged adults. Researchers reviewed studies that measured the association between participating in exercise for 12 or more months and cognitive function in adults aged 18 to 50 years. Seven studies found a significant positive correlation between higher levels of physical activity and improved executive function. Three of four studies found higher levels of exercise significantly improved specific aspects of memory function. And two of seven studies found a positive association between higher levels of physical activity and enhanced processing speed. Furthermore, researchers showed that the more time adolescents spent in moderate or moderate-to-vigorous physical activity, the greater their attention span.

Yoga also acts as a cognitive enhancer. Scientists examined the effects of yoga on emotional intelligent quotient (EQ) in 72 business executives, a population known to experience excessive stress. The yoga intervention resulted in a 72% increase in EQ.

In another study, researchers investigated the effects of Hatha yoga in 118 community-dwelling seniors with a mean age of 62 years. The subjects were randomly divided into two groups. In the eight-week study, one group performed Hatha yoga during hour-long classes three times per week while the other group participated in stretching-strengthening exercises as a control. The researchers measured the subjects’ executive function using established tests. At the study’s termination point, compared with subjects who performed stretching-strengthening exercises, subjects performing yoga had marked improvements in executive function measures including working memory capacity and efficiency of mental set shifting and flexibility.

### NUTRACEUTICAL SUPPORT FOR BRAIN HEALTH

Several botanicals and nutrients have been shown to enhance cognitive function.

**Bacopa Monnieri**

*B. monnieri* is a botanical known for its memory-enhancing effects. A growing number of studies indicate bacopa is a potent cognitive enhancer in individuals of all ages. One of the earlier
studies of bacopa investigated its effects on cognitive abilities in healthy humans aged 18 to 60 years old.\textsuperscript{48,49} In this double-blind, placebo-controlled, randomized study, participants received 320 mg per day of a bacopa extract or placebo daily for 12 weeks. Bacopa significantly enhanced speed of early information processing, verbal learning rate, and memory consolidation compared to placebo. The researchers concluded that bacopa “may improve higher order cognitive processes that are critically dependent on the input of information from our environment such as learning and memory.”

Another randomized, double-blind, placebo-controlled trial published a year later investigated the effects of bacopa extract in 76 adults between 40 and 65 years old.\textsuperscript{50} In this study, bacopa significantly improved the subjects’ ability to remember new information.

More recent studies have yielded equally promising findings. Researchers conducted a randomized, double-blind, placebo-controlled, 12-week trial that studied the effect of 300 mg per day of bacopa extract on subjects aged 65 or older.\textsuperscript{51} The study included 48 subjects without dementia who were randomly divided to receive either bacopa or a placebo. The researchers measured the delayed recall score from the Rey Auditory Verbal Learning Test (AVLT). They also evaluated other cognitive measures including the Stroop Task, which measures the ability to discount irrelevant information, the Divided Attention Task (DAT), and the Wechsler Adult Intelligence Scale (WAIS), which is used to assess immediate working memory. Anxiety, depression, and mood were also evaluated in the subjects.

After controlling for baseline cognitive dysfunction, the researchers found that subjects given bacopa had better delayed word recall memory scores compared to subjects taking placebo. For the Stroop Test, the bacopa group improved while there was no change in the placebo group. Depression and anxiety scores also fell over time in the bacopa group while increasing in the placebo group.

Bacopa has also demonstrated beneficial effects in attention deficit hyperactivity disorder (ADHD). In 31, 6- to 12-year-old children with ADHD, six-month administration of 225 mg per day of bacopa extract was associated with a reduction in ADHD symptoms, with the exception of social problems.\textsuperscript{52} Bacopa led to a 93% reduction in symptom scores for restlessness and an 89% improvement in self-control. Attention-deficit symptoms became less frequent in 85% of the children. Symptom scores for learning problems, impulsivity, and psychiatric problems declined in 78%, 67%, and 52% of the children, respectively. In total subtest scores for ADHD symptoms, 74% of the children had up to a 20% reduction, while the remaining 26% showed a 21% to 50% decline.

Additionally, animal studies indicate bacopa is an adaptogen that can protect against chronic stress,\textsuperscript{53,54} suggesting it has the potential to inhibit the detrimental effects of stress on the brain.

**IODINE**

Iodine is a mineral essential for cognitive health and the production of thyroid hormones. One analysis of the medical literature found that iodine-deficient children and adults had a mean IQ score 13.5 points lower than people who were not iodine deficient.\textsuperscript{55} Another meta-analysis examined the effects of iodine on the IQ of children in China.\textsuperscript{56} Compared to children whose mothers were iodine deficient, children born to mothers who supplemented with iodine during pregnancy had IQs that on average were 8.7 points higher. For children born more than 3.5 years after an iodine supplementation program was introduced, IQs were 12 to 17.25 points higher. Severely iodine deficient children had an average IQ loss of 12.45 points.

**IRON AND OMEGA-3 POLYUNSATURATED FATTY ACIDS (PUFAS)**

Two other nutrients important to cognitive function include iron and omega-3 PUFAs. Worldwide, iron deficiency is the most widespread nutritional deficiency and has been associated with cognitive impairment in numerous studies. Researchers evaluated the effect of supplemental iron in a study of 200 female high school students who were randomized to receive either 50 mg of iron (as ferrous sulfate) twice a week for 16 weeks, or no supplementation.\textsuperscript{57} Administration of iron enhanced the attention span of the subjects.

In another study, scientists evaluated the prevalence of iron deficiency in anemic and nonanemic adolescents and measured the effect of iron deficiency on cognitive function in both groups.\textsuperscript{58} In both anemic and nonanemic iron-deficient girls, scholastic performance, IQ, scores of mental balance, attention and concentration, verbal memory, and recognition were all lower compared to a control group of girls who were not iron deficient.

Omega-3 PUFAs also have a beneficial effect on brain health. When combined with B vitamins, omega-3 PUFAs were found to slow the rate of brain atrophy in elderly subjects.\textsuperscript{59} Additionally, omega-3s have been shown to enhance school performance and reading comprehension in 8- to 11-year-old children.\textsuperscript{60} Furthermore, a randomized, placebo-controlled trial demonstrated that supplementation with omega-3 PUFAs can reduce the verbal memory impairment that occurs over time in individuals who suffer from loneliness.\textsuperscript{61} Finally, when combined with aerobic exercise, omega-3s have been shown to inhibit the decline of gray matter volume and circulate cortex atrophy in patients with mild cognitive impairment.\textsuperscript{62}

**CORRECTING EPIGENETIC DEFECTS**

Because epigenetic processes may contribute to cognitive impairment, a cognitive-enhancing program should include supplements that restore epigenetically modified genes. Epigallocatechin gallate (EGCG) from green tea is an epigenetic modulator.\textsuperscript{63} A human study found green tea consumption resulted in a decline in DNA methylation in persons with gastric cancer.\textsuperscript{64} EGCG is also a histone modifier.\textsuperscript{65,66} Furthermore, green tea restores the activity of genes that play a role in cancer inhibition after these genes have been epigenetically silenced.\textsuperscript{67} Green tea also has cognitive-enhancing effects. When combined with L-theanine, it enhances memory and attention in individuals with mild cognitive impairment.\textsuperscript{68} Furthermore, EGCG has been shown to improve cognitive function in mice and humans with Down’s syndrome.\textsuperscript{69}

Folate supplementation is another means of protecting against epigenetic defects. Folate modulates DNA methylation by generation of the methyl donor S-adenosylmethionine (SAMe).\textsuperscript{70} The enzyme methylenetetrahydrofolate reductase (MTHFR) is required to generate SAMe. However, many individuals have a defect in MTHFR that results in lower enzyme activity.\textsuperscript{71} This is associated with reduced methylation capacity.\textsuperscript{72} Supplementing with L-5-methyltetrahydrofolate (L-5-MTHF) instead of folic acid can improve folate metabolism in people who have this defect.

**A BOOST TO BRAIN HEALTH**

Many factors are involved in memory impairment in young and middle-aged adults as well as in adults with normal, age-related cognitive decline. However, research clearly indicates that cognition can be improved in all age groups through the use of lifestyle alterations such as weight loss, adequate sleep, stress reduction, and increased exercise, as well as the use of nutritional supplements such as bacopa, iodine, omega-3 fatty acids, iron, green tea, and...
Concerns over the health effects of electromagnetic radiation date back to the World War II era when expanded use of radar and radio waves led to scattered reports of fatigue, headaches, loss of libido, and other symptoms following exposure to EMFs. Only in recent decades, however, have well-designed epidemiological studies sought to methodically investigate the potential harmful consequences of EMF exposure. The endeavor is fraught with difficulty for a variety of reasons including problems quantifying EMF exposure levels and the long latency period between exposure and some of the purported disease manifestations of EMFs, like cancer. Nevertheless, a sufficient body of research now suggests a probable link between EMFs and increased risk for acute and chronic health problems.

DIFFERENT TYPES OF EMFS

EMFs can be broadly categorized according to their position along the electromagnetic spectrum. EMFs on the lower-frequency end of the spectrum include extremely low frequencies (ELFs), radio-frequencies (RFs), microwaves, and infrared. All of these EMFs are classified as nonionizing types of radiation.

EMFs on the higher-frequency end of the spectrum include ultraviolet, X-rays, and gamma rays. These EMFs are considered ionizing because they carry sufficient energy to remove electrons from the orbitals of atoms, thereby ionizing them. Visible light lies between these two ends of the spectrum, red light bordering on the lower-frequency infrared end and violet on the higher-frequency ultraviolet end.

Adverse health effects from excessive exposure to ultraviolet (UVA and UVB), X-rays, and other forms of ionizing radiation have been well-documented. This article will focus on the more
ELFs for periods up to 20 years and found no disruptive effect on these hypotheses are not without their critiques. Some studies, for example, the spin dynamics of electrons in free radicals, which may propose ELFs may cause subtle changes in biological processes, properties.12

A tremendous amount of research has investigated the effects of ELF radiation on human health, most of it centering around its association with cancer. But a growing number of reports also implicate ELFs in the development or exacerbation of other types of conditions such as neurological and developmental disorders.

ELFS AND CANCER
Research over the last 30 to 40 years has established a strong epidemiological link between ELF exposure and increased risk for developing certain types of cancer, especially leukemia. The highly suggestive data led the World Health Organization’s International Agency for Research on Cancer to formally designate ELF radiation as “possibly carcinogenic to humans (Group 2B)” in 2002.8

Despite the compelling epidemiological evidence, however, researchers have been hard pressed to find a plausible mechanism to explain how ELFs affect biological systems. Physical laws argue that field strengths in the ELF range are far too low to ionize atoms or molecules, induce a thermal effect, disrupt chemical bonds, or in any other way adversely affect human cells or tissues.8,9 But some propose ELFs may cause subtle changes in biological processes, such as the spin dynamics of electrons in free radicals, which may influence free radical concentrations and activity, increasing their potential to cause damage in the body.10 Others believe ELFs, through undetermined means, suppress endogenous production of melatonin,11 a hormone with antioxidant and possible anticancer properties.12

These hypotheses are not without their critiques. Some studies, for example, have examined residential and occupational exposures to ELFs for periods up to 20 years and found no disruptive effect on melatonin production.13,14

LEUKEMIA
Much of the research on ELFs and cancer has focused on a correlation between residential exposure and increased risk of developing leukemia. Researchers first reported such an association in 1979 in the American Journal of Epidemiology. The investigators carried out a case-control study in the greater Denver area and found a marked elevation in the number of childhood and adult leukemia cases among subjects with higher residential exposure to ELFs from power lines.15

Since publication of this seminal paper, over twenty additional studies have been conducted, most corroborating the Denver findings.9 Five separate meta-analyses, where researchers analyze the results of a group of studies, have also been published supporting a link between ELF exposure and increased leukemia risk, especially in children.16–20 A consistent finding among these meta-analyses is that the health impacts of ELFs are dose dependent—i.e., risk level rises as exposure increases.

ELF dosing is typically expressed in microtesla (µT) or milligauss (mG) units, where 1 µT = 10 mG. Most people experience average daily levels of 0.1 to 0.2 µT at home or at the workplace.21,22 Exposure levels of >0.4 µT are relatively rare.16 In a 2000 meta-analysis published in the British Journal of Cancer, researchers reported no significant risk effect of ELFs at exposure levels of <0.4 µT, but a significant doubling of childhood leukemia risk with exposures of ≥0.4 µT.16

Similarly, another group of researchers found little to no risk associated with exposure levels of 0.1 to 0.3 µT, but they did find an elevated risk for exposures >0.3 µT.17 Other groups of scientists have reported increasing risk as ELF exposure levels increased from 0.1 to 0.4 µT and higher.18,19 These findings provide consistent evidence of an increased risk of leukemias, especially childhood leukemia, with increasing exposure to ELF radiation.

LYMPHOMA
A less robust, but nonetheless significant, association has been established between ELF exposure and risk for childhood lymphoma. A meta-analysis examined epidemiological data from five studies and, among those reporting dose-response data, found a significantly higher incidence of lymphoma in children with the highest residential exposure to ELFs from power lines.2 Risk appeared to be greatest for Hodgkin’s disease (a form of lymphoma).23

BREAST CANCER
A considerable amount of research has attempted to shed light on the relationship between ELF exposure and breast cancer. Early trials yielded variable results, some reporting evidence of a positive correlation,24,25 others finding a weak or no association.26,27 The data inconsistencies have been attributed to poor study design, widely varying methodologies, and frequent use of proxy instead of direct measurements of ELF exposure.28,29

In 2010, a meta-analysis examining data from 15 case-control studies from 2000 to 2009 reported no relation between incidence of breast cancer and ELF exposure.29 Three more recent meta-analyses, however, arrived at different conclusions. One group of researchers evaluated 23 case-control studies and found a small, but significant elevation in overall risk for breast cancer among women with higher exposure to ELFs. Analyzing members of different subgroups revealed that women in their premenopausal years, those with estrogen receptor-positive status, and those who experienced ELF exposure from multiple sources had the highest risk.10

Other scientists likewise observed an increase in breast cancer risk for women in their premenopausal years who had higher ELF exposure.31 Researchers also pooled data from 18 studies and found a significant elevation in risk among men with higher ELF exposure.28 Thus, while not yet conclusive, most of the evidence from several decades of research appears to support an association between ELF exposure and an increased risk for breast cancer.

BRAIN TUMORS
Over the years, scattered reports have appeared in the medical literature implicating ELFs in the development of tumors of the brain and/or nervous system.15,32 Compared to the strong link established between ELFs and leukemia, however, the evidence regarding brain cancer is relatively weak.

In 2010, researchers conducted a meta-analysis of studies from 1988 to 2010 and found no statistically significant association between brain cancer and any measure of residential ELF exposure. Although some of the calculated risks for exposures of ≥0.4 µT appeared to be elevated, these measures did not reach statistical significance,
and the overall pattern of risk across increasing exposure categories was not indicative of a dose-response relationship. These findings corroborate the results of an earlier meta-analysis, although the authors of that study concluded a moderate increase in the risk of brain cancer at higher exposure levels of >0.3 or 0.4 µT could not be ruled out.

OTHER CANCERS
A few studies have reported associations between ELF exposure and cancers of the biliary ducts, larynx, liver, lung, male genitalia, and skin. In general, the associations are weak, but one group of researchers found a median fourfold increase in risk of nonseminoma testicular cancer among men 40 years of age or younger with the highest occupational exposure to ELFs.

ELFS AND OTHER DISORDERS
Some researchers believe ELFs may play a role in other, noncancer types of health conditions. For example, researchers found an association between ELF exposure among electric utility workers and mortality from acute myocardial infarction and arrhythmia-related conditions. Other scientists have linked high occupational exposure to ELFs to an increased risk for neurodegenerative disorders such as amyotrophic lateral sclerosis (ALS) and Alzheimer’s disease. Additional research is needed to confirm or refute these findings.

RADIOFREQUENCY (RF) EMFS
Although there is no formal definition of RF EMFs, a convenient categorization places them above ELFs and below infrared on the electromagnetic spectrum (~30 kHz to 300 GHz). RF EMFs include fields generated by radio and television broadcast towers, cell phones, cordless phones, smart meters, wi-fi networks, wearable smart devices, and microwave ovens. Most of the controversy and debate over the health effects of RF EMFs centers around its carcinogenic potential, but exposure to RFFs can also pose a danger to persons with implanted cardiac devices and may entail additional health risks.

RF AND CANCER
Research investigating the effects of RFs on health has focused primarily on cell phone use and the incidence of brain cancer. Although the results of this research are conflicting, some studies provide compelling evidence of a connection between longer-term use of cell phones and higher risk for particular types of brain tumors. On the strength of this evidence, the World Health Organization’s International Agency for Research on Cancer issued a press release in 2011 assigning RF EMFs to the same risk category as ELF EMFs – i.e. “possibly carcinogenic to humans (Group 2B).”

As with ELFs, research implicating RFs in the development of cancer is primarily epidemiological in nature. Mechanistic explanations of how RFs impact biological systems are still elusive and highly controversial. A number of scientists and research agencies firmly maintain that no candidate mechanism proposed thus far has been experimentally validated or shown to operate in human biological systems at levels of exposure found in the everyday environment.

A growing number of studies, however, report evidence of increased free radical activity and oxidative stress in the cells and tissues of animals and humans following RF exposure.

BRAIN CANCER
Of all the health impacts of EMFs, perhaps none has aroused more interest or controversy than the potential link between cell phones and brain cancer. Cell phone use has exploded in recent years, worldwide mobile subscriptions now surpassing 7 billion. Given the near universality of cell phone use, even a small increase in cancer risk would have enormous public health implications.

Many studies over the last two decades have attempted to answer the question, “Does using a cell phone increase the risk of brain cancer?” The results of these studies are conflicting. In 2008, a meta-analysis examined data from nine case-control studies and found no evidence of increased risk for total brain cancer or brain cancer subtypes among cell phone users of less than 10 years. Users of 10 or more years showed a slightly elevated risk of total brain cancer, a phenomenon the authors speculate may relate to use of older, higher-powered analog cell phones. Another 2008 meta-analysis of 19 case-control studies reported a significantly elevated risk for ipsilateral (same side as phone use) glioma and ipsilateral acoustic neuroma among users of cell phones for 10 or more years.

In the following year, another group of researchers pooled data from 23 case-control studies and, once again, found evidence linking cell phone use of 10 or more years to increased overall risk for brain tumors. These researchers also noted that when studies were categorized according to blinding status (an indicator of study quality), a mild increase in brain cancer risk emerged even in cell phone users of less than 10 years.

In contrast to the previous reports, a 2012 meta-analysis (which was funded in part by the telecommunications industry) concluded that the totality of evidence did not support a link between cell phone use and any type of brain cancer. Although they found elevated risks for glioma, acoustic neuroma, and meningioma in long-term cell phone users, these values were determined to be nonsignificant. These researchers also reviewed the literature on cordless phone use and incidence of brain cancer and found widely divergent results, some studies reporting no increase in risk, others finding a substantially increased risk. Since this meta-analysis, several additional papers have been published either affirming or refuting a connection between cell phone use and brain cancer risk.

Thus, after almost 20 years of investigation, a substantial amount of data has amassed that does not allow us to draw definitive conclusions, but nonetheless provides sufficient evidence for concern over long-term use of cell (and possibly cordless) phones and increased risk of developing certain types of brain cancer.

RF AND OTHER DISORDERS
A variety of noncancer health effects have been attributed to RF EMFs from devices such as cell phones, microwave ovens, smart meters, and wi-fi equipment. The scope of reporting on this topic is so large as to preclude a detailed analysis, but some of the better-supported associations can be mentioned.

In 2015, scientists summarized research dating back to the 1970s linking RF exposure to neuropsychiatric effects. Symptoms reported in 10 or more studies (and thus unlikely to have been caused by chance) included sleep disturbance, headache, fatigue/tiredness, depression, sensory changes, and cognitive dysfunction. These symptoms were associated with RF exposure from cell phones, cell phone base stations, smart meters, and television and radio broadcast facilities.

In addition to neuropsychiatric symptoms, an association between speech problems in children and maternal use of cell phones (but not cordless phones) was recently reported by an Iranian research team. The investigators cited supportive studies showing neuropsychology can be induced in mice by exposing them to RF radiation in utero.

Finally, it has been fairly well documented that RF radiation from microwave ovens, cell phones, electrical dental devices, magnetic resonance induction (MRI) equipment, and other sources can
interfere with the proper function of cardiac implantable electronic devices (CIEDs) such as pacemakers and defibrillators. Although newer CIED designs incorporating titanium casing, signal filtering technology, smaller battery size, and other improvements have substantially reduced the risk of outside electromagnetic interference, persons with CIEDs should still use caution around sources of RF radiation and discuss with their doctors which RF-emitting devices are safe and which may pose a potential danger.

NATURAL APPROACHES TO EMF PROTECTION

Although the data presented above do not conclusively link EMFs to any particular disease or disorder, they do validate concerns about its potential negative health impacts. Reducing the health risks posed by EMFs can be accomplished by limiting exposure to ELF and RF radiation to the greatest extent possible. While it may be impractical to try and completely avoid EMFs, even simple steps such as reducing cell phone use, moving electronic clocks, radios and other devices away from the bed at night, and avoiding sleeping under an electric blanket can substantially reduce EMF exposure. Persons who live in close proximity to high-voltage power lines or have occupational exposure to EMFs may face a more difficult decision about whether to relocate or change jobs.

Anyone concerned about excess EMF exposure may also wish to consider taking antioxidant supplements to increase bodily resistance to oxidative stress. As discussed earlier, increased production of oxidative free radicals is one of the mechanisms by which EMFs are believed to damage cells and tissues. Nutrients with well-documented antioxidant activity include vitamins C and E, beta-carotene and other carotenoids, alpha-lipoic acid, coenzyme Q10 (CoQ10), melatonin, and polyphenolic compounds. The body also requires zinc, copper, manganese, selenium, and N-acetyl-L-cysteine (NAC) to synthesize important endogenous antioxidant systems. Botanicals rich in antioxidant compounds include green tea, Ginkgo biloba, and turmeric. Studies show most of these nutrients and botanicals enhance the body’s antioxidant status and effectively reduce markers of oxidative stress in human cells and tissues following radiation exposure. Of note, beta-carotene, alpha-lipoic acid, and Ginkgo biloba extract were used following the Chernobyl nuclear accident in the former Soviet Union and were shown to significantly lower oxidative stress markers and the activity of mutagenic agents in radiation-exposed adults and children.

MOUNTING CONCERNS

With the ever-increasing use of electronic devices and expanded access to electrical power across the globe, concerns about the health impacts of EMFs continue to mount. Several decades of research have probed the connection between EMF exposure and adverse health effects. While the results have been largely inconsistent, there is credible evidence linking EMFs to a number of health conditions, including certain types of cancer. The association is particularly strong for heavy exposure to ELF’s from power lines and increased incidence of childhood leukemia, and for long-term exposure to RFs from cell phones and higher risk for some types of brain tumors.

In order to minimize health risks associated with EMFs, exposure should be limited to the greatest degree possible. Studies also suggest antioxidant supplementation might be effective at reducing oxidative stress in the body and mitigating some of the damaging effects of radiation.

REFERENCES:
[Article in Chinese; abstract in English.]

DID YOU KNOW?

- Pregnenolone inhibits midbrain dopamine activation, which plays a role in addiction to many types of drugs, suggesting that balancing levels of this hormone may help reduce cravings for addictive drugs.

- Inflammatory bowel disease is associated with an increased risk of bone loss due to such factors as inflammation, use of corticosteroids, and vitamin D deficiency.

- In a study of high school students, vitamin C supplementation reduced anxiety levels.

- Ashwagandha (Withania somnifera) supplementation is associated with significant increases in muscle mass and strength, and may be useful in conjunction with a resistance training program.

- Use of antidepressants—specifically of selective serotonin reuptake inhibitors (SSRIs) by women during pregnancy, particularly in the second and/or third trimesters—is associated with an increased risk of autism spectrum disorder in their children.

- Four months of supplementation with cinnamon, chromium, and carnosine decreased fasting plasma glucose and increased fat-free mass, but not body weight, in overweight or obese prediabetic subjects.

- Resistance-trained females given the amino acid citrulline (as citrulline malate) experienced enhanced performance in upper- and lower-body resistance exercise and less perceived exertion.

- In a rat model of ischemic stroke, intraperitoneal progesterone given in doses of 8, 16, or 32 mg/Kg resulted in improvements in spatial and long-term memory as well as reductions in gait impairments. Doses of 8 and 16 mg/Kg were associated with smaller infarct volumes.

- Sleeping less than seven hours per night is associated with increased distracted eating or drinking beverages other than water, including sugar-sweetened beverages, while watching television or participating in another activity.

- Berberine can help restore a regular menstrual cycle and enhance the ovulation rate in some women with polycystic ovary syndrome (PCOS). In normal-weight women with PCOS, berberine may also lower sex hormone binding globulin, insulin resistance, total cholesterol, triglycerides, and low-density lipoprotein (LDL) cholesterol.

- Cadmium is a component of cigarette smoke that damages ovarian oocyte follicles. Blueberry extract protects against cadmium ovarian toxicity in mice.

- Laboratory data suggest green coffee bean extract exerts antibacterial activity against periodontal pathogens.

- Having a higher body mass index (BMI) and greater body fat percentage is associated with lower vitamin D levels in people aged 65 and older.

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